CORRESPONDENCE

Postpartum changes in maternal blood lead concentrations

Sir,—Ernhart and Greene (1992;49:11–13) cite a publication of mine1 as the only report in the literature suggesting that an increase of blood lead concentration occurs during pregnancy. This is not accurate. Thompson et al2 described a case of a woman who experienced abdominal cramps during several pregnancies and ensuing periods of lactation. Three months after her last delivery, her blood lead concentration was 74 μg/dl and that of her infant was 55 μg/dl. The authors noted that she had lead encephalopathy at the age of 18 months and speculated on the possibility that increased turnover of calcium during pregnancy and lactation had released lead stored in bone. Taken together our two reports make a strong case for increased blood lead concentrations during pregnancy.

I attempted some years ago to examine the matter further by following up a husband and wife's blood lead concentrations and isotope ratios before her conception and during her pregnancy, but the study was spoilt by their having been exposed to a foreign source of lead during her pregnancy and by some lead concentration measurements having been lost due to a procedural error in the laboratory. As the results have some bearing on Ernhart and Green's paper, however, I present them here. The husband was the same that I had followed up previously, but as he had remarried, his wife was new to the study. In May 1983, they spent a month in Honduras and on their return the wife spent six weeks in the western United States. She conceived in December 1983 and they returned to Honduras for the months of May and June 1984. She delivered on 15 September. Blood lead concentration and isotope ratios were obtained for the periods they were in Dallas and the cord blood was analysed. Unfortunately, all concentration measurements for her last trimester failed.

From the figure it is apparent that their blood lead isotope ratios were at first rapidly changing. The reason may be due to their having been exposed to lead from sandpapering old paint in their home. On their return from their first trip to Honduras their blood lead concentrations had increased and a change in isotope ratio occurred, considerably so in the case of the wife. From the magnitude of the changes, it is likely that the 209Pb/207Pb ratios in the environment of Honduras were in the range 1.18 to 1.19. Over the next 10 months, which includes the first five months of the wife's pregnancy, their blood lead concentrations decreased. So did the husband's isotope ratios, whereas those of his wife remained steady. The second two months spent in Honduras brought about an increase of 2 μg/dl in each of their blood lead concentrations which translates into a 20% increase in the case of the husband, and a doubling in the case of the wife. This considerable change brought about a correspondingly large change in her isotope ratios. During her last trimester, these rapidly changed back towards her Dallas values. The lead concentration in the cord blood was 2·0 μg/dl and it may be assumed with confidence that her blood lead at delivery was 25% higher, or 2·5 μg/dl, which is identical to her blood lead concentration before she left for Honduras.

Although the data are incomplete, they carry sufficient information to negate the hypothesis that a rise in blood lead concentration is a necessary consequence of pregnancy. In this regard, they agree with Ernhart and Greene’s findings, but the 1 μg/dl decrease in the wife’s blood lead concentration would seem to reflect an overall decrease in exposure rather than a change in haematocrit as her husband’s blood lead concentrations decreased over the same period. If my two studies, the findings of Thompson et al, and Ernhart and Greene’s data are considered together the conclusion to be drawn is that some but not necessarily all women release measurable amounts of lead from bone during pregnancy. The question needs to be answered as to whether this reflects a particular physiological disposition or whether it reflects only those with high stores accumulated in childhood. Obviously, the number of women with large stores is far smaller than it was 20 years ago. Ernhart and
Greene have shown that on average the blood lead concentration of their sample rose post partum, but a more appropriate question to have asked would be, how many out of that group showed the reverse? This figure would be an indication of how many women today in the United States are experiencing increased blood lead concentrations during pregnancy.

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An industrial disease

Sir,—The Industrial Injuries Advisory Council is again considering whether chronic bronchitis and emphysema in coal miners should be prescribed as industrial diseases for which benefit may be paid. This is welcome news, but any benefit miners might receive will be too little and too late.

Coal miners in the past who experienced excessive exposure to nitrous fumes from shortfiring activities underground have been eligible for benefit and should have qualified for prescribed disease “poisoning by nitrous fumes” (PD C15 now termed PD C15). The poisoning may be acute or chronic but occasionally acute and chronic poisoning may affect the same man. Miners claiming benefit for chronic poisoning have invariably been turned down by medical boards, however, unless there was also evidence of a dramatic episode of acute poisoning! The requirement of previous acute poisoning to qualify for acceptance as a case of chronic poisoning is absurd. Surely no case of chronic lead or chronic carbon monoxide poisoning would be rejected for benefit in the absence of acute disease?

At the 1936 conference in Cardiff on “lung trouble in the anthracite collieries” many medical experts noted the relation between chronic fume exposure and chronic bronchitis and emphysema. John Craw who attended the conference was so convinced of the relation for both south Wales coal miners and in his Cumbrian haematite ore miners that he organised the near elimination of the fume hazard in the Cumbrian mines. Some 40 years later he was able to report the virtual eradication of chronic bronchitis and emphysema and his concomitant programme of dust suppression also resulted in the elimination of pneumoconiosis. This surely must be a classic example of industrial medicine, with the identification of the causes (fume and dust) of the diseases (emphysema and pneumoconiosis) and their elimination by preventive measures.

In considering claims for PD C15 the medical boards have obviously been influenced by the strong relation between cigarette smoking and chronic bronchitis and emphysema. I wonder whether the cumulative effect of nitrous fumes from both industry and tobacco smoke and the importance of coal and carbon dusts as carriers of fume have been fully appreciated. The reasons and evidence for my views have been published previously.4–7 The clinical picture of nitrous fume poisoning as described in the DSS pamphlet (NI 229 September 1990) notes on the diagnosis and claims for industrial scheme benefits is ambiguous and needs rewriting.

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1 Jones TD. Silicosis in the south Wales Coalfield—Part 1. Lung trouble in the anthracite collieries. Proc South Wales Institute of Engineers 1936; 52:157–244.